Neurol Sci (2008) 29:219–227
DOI 10.1007/s10072-008-0971-9

ORIGINAL ARTICLE

Recognition of emotions from visual and prosodic cues in Parkinson's disease

Alessandra Ariatti · Francesca Benuzzi · Paolo Nichelli

Received: 2 March 2008 / Accepted in revised form: 3 July 2008

Abstract *Objective* To assess whether Parkinson Disease (PD) patients are impaired at perceiving emotions from facial and prosodic cues and whether any putative defective performance concerns recognition of a particular emotion. *Background* Braak et al. [1] demonstrated that in different stages PD pathology involves the nigrostriatal system, the amygdala, and the insular cortex. Discrete brain lesions to these structures can cause selective deficits in recognising facial and prosodic stimuli expressing particular emotions. However, the investigation of facial and prosodic emotional processing in PD patients has lead to conflicting results. *Materials and methods* We compared 27 cognitively unimpaired PD patients with control subjects by means of the Facial Emotion Recognition Battery and the Emotional Prosody Recognition Battery. *Results* PD patients were impaired in recognising, selecting, and matching facial affects. In particular, the Facial Emotion Recognition Battery demonstrated a severe impairment in recognising sad and fearful faces. In the Emotional Prosody Recognition Battery PD patients demonstrated a diffuse impairment, including the recognition of emotional and propositional prosody. *Conclusions* Face emotion processing is impaired in PD patients, with a disproportionate deficit involving fear and sadness. The pattern of face expression processing impairment in PD patients might depend on the regional distribution of the pathology. The widespread involvement of both emotional and propositional prosodic processing parallels the aprosodic characteristics of Parkinsonian speech production.

Keywords Parkinson's disease · Emotions · Facial expressions · Emotional prosody

A. Ariatti · F. Benuzzi · P. Nichelli (✉)
Department of Neurosciences
University of Modena and Reggio Emilia
Nuovo Ospedale Civile Sant'Agostino-Estense
Via Giardini, 1355
41010 Modena, Italy
e-mail: nichelli@unimo.it

Introduction

Both neuropsychological and neuroimaging data support the notion that cortical and subcortical regions are involved in processing emotions form facial and prosodic cues. A large number of different structures participate in recognising the facial expression of emotions: among them, the occipito-temporal cortex, the amygdala, the orbito-frontal cortex, the basal ganglia, and the right parietal cortex [2]. In contrast, studies on emotional prosody do not clearly indicate any similar neural network. In general, recognising emotions from prosody alone is more difficult than recognising emotions from facial expressions. Certain emotions, such as disgust, can be recognized only very poorly from prosody. The recognition of emotions from the voice draws on multiple prosodic cues, which are, in turn, processed by systems that in part are neuroanatomically segregated towards one or the other hemisphere. In numerous studies, the right fronto-parietal region has consistently emerged as critical for recognising emotional prosody [3–6], a role it might play in association with the basal ganglia [4].

Specific cerebral structures seem to be involved in the processing of specific visual and prosodic emotions. Neuropsychological [7, 8] and neuroimaging data [9] support this view. For instance, there is a wide agreement

that amygdala mediates the recognition of fear across visual, auditory and gesture stimuli [10].

Functional imaging studies demonstrate the involvement of the basal ganglia, but also of the insular cortex, in facial disgust recognition [11–14]. The role of these structures in disgust processing is also supported by studies that have found that individuals with neurodegenerative diseases are impaired at recognising the facial expression of disgust. For instance, patients with symptomatic Huntington's disease [15–18] and pre-symptomatic Huntington's disease gene carriers [11, 19] showed an overall deficit in recognising emotions, with a particularly severe deficit for recognising expressions of disgust from both face and voice. Patients with Wilson's disease [20], Tourette syndrome with comorbid obsessive compulsive behaviour, and people with obsessive compulsive disorder [21], all show selective deficits in facial disgust recognition.

In contrast, it is far from clear whether Parkinson's Disease (PD) patients are impaired at perceiving emotional facial affects. Individuals with PD have a reduced ability in making spontaneous emotional expressions and have monotonous, flat, and poorly inflected speech. It has been suggested [22] that there is a strong link between perception and expression of emotions. Jacobs et al. [23] demonstrated that PD patients are impaired in imaging, perceiving, and expressing emotional faces. They explained the relationship between motor and perceptual-imaginative aspects with the notion of a central processor located in the basal ganglia or dependent upon neostriatal function. Indeed, the neostriatum receives input from the whole cortex, including projections from the inferior and the superior temporal gyri, which are involved in visual perceptual representations of face and facial emotions. Dujardin et al. [24] established that early in the course of PD, non-verbal emotional information processing is disturbed and that untreated PD patients are significantly impaired in decoding emotional facial expressions. They suggested that in PD nigrostriatal dopaminergic depletion leads not only to motor and cognitive disturbances, but also to emotional information processing deficits. Specifically this study showed that PD patients were less accurate than controls in decoding anger, sadness and disgust from facial expressions, regardless of the expression's intensity level (low or mild). Suzuki et al. [25], using the facial expression hexagon, showed that PD patients were selectively impaired at recognising the facial expression of disgust. Wieser et al. [26] found that PD patients report less arousal compared to healthy controls during extended viewing of emotional pictures. They demonstrated that reduced arousal was not attributable to generally increased fatigue or an overall enhanced depression score.

Tessitore et al. [27] explored the neural basis of abnormal emotional behaviour in PD using fMRI. They found that, in non-medicated PD patients, an emotional task was not associated with bilateral amygdala activation as in normal subjects. Dopaminergic repletion, however, was shown to restore this response, at least partially. Absence of amygdalar response in PD patients was also found by Yoshimura et al. with visual event-related potentials (ERPs) related to viewing of fearful facial expressions [28].

Sprengelmeyer et al. [29] investigated the effect of dopamine medication and observed impaired recognition of disgust and anger from facial expressions. This deficit was more severe in non-medicated patients than in medicated patients with PD. The dopaminergic role on emotion perception and recognition was also emphasized by Salgado-Pineda at al. [30].

In contrast with this hypothesis, several studies failed to demonstrate any difference between patients and controls in facial emotion tasks [31-33]. Conflicting results have been also reported by studies investigating defective production and recognition of emotional and propositional prosody (e.g. the prosody involved in asking a question or stating an affirmation [34]) in PD subjects [33, 35–39]. Caekebeke et al. [40] demonstrated a deficit in producing, but not in recognising, prosody and facial expression, thus suggesting that the origin of the impairment may be the result of dysarthria and not necessarily related to defective encoding/decoding of affective behaviour. Kan et al. [41] found that PD patients are impaired in recognising fear and disgust from facial expression, but they did not find significant differences between PD patients and controls in recognition of emotion from prosodic and written verbal stimuli. Yip et al. [42] evaluated the emotion recognition abilities of a group of bilateral and right-sided PD patients. Results demonstrated that although patients with bilateral PD were impaired in recognising all six basic emotions, fear, followed by sadness, was more impaired than other basic emotions (disgust, surprise, anger and happiness). Bilateral PD patients showed defective performance in all tests used, irrespective of the modality and the type of experimental task. In contrast, patients with right-sided PD had difficulties with facial emotion identification and prosodic emotion identification only. Furthermore, the recognition of all emotions in patients with right-sided PD was significantly impaired and particularly the recognition of sadness and disgust.

Summing up, there is no clear evidence of either a general or a specific impairment of emotional processing in PD. When present, the impairment seems to involve negative facial affects such as disgust or fear. No specific deficit for prosodic emotion has been found. The present study addressed this issue evaluating the presence of a general or selective emotional impairment in PD across the visual and the auditory domain. We set up two batteries for the facial and the prosodic emotion evaluation using equivalent tasks within the two batteries. Our results suggest a specific impairment in recognising fear and sadness from facial cues in PD patients. In addition,

we found defective performances in PD patients both in emotional and non-emotional prosody processing.

Materials and methods

Subjects

Patients with diagnosis of idiopathic Parkinson Disease (PD patients) were selected through the outpatient Movement Disorder Clinic of the Modena University. They included 27 PD patients (15 males and 12 females). All patients had a negative CT or MR scan of the brain. With respect to the most affected body side, 13 patients were considered right-sided and 13 left-sided. This classification was not applicable for one patient (V.M.). Sixty-eight healthy volunteers (28 males and 40 females; mean age= 62.5 years) participated in the study as controls. All participants were free of alcoholism, psychiatric illness, head trauma, cerebrovascular disease, and other severe neurological conditions.

Criteria of patient selection included absence of general and severe cognitive deterioration (MMSE ≥23) [43], severe depression as evaluated by the Beck Depression Inventory (BDI <23 [44]) and obsessive-compulsive disorder assessed by the reduced version of the Maudsley Obsessional-Compulsive Questionnaire (MOCQ-R) <75 percentile [45]. All subjects were fully independent in everyday life activities and they all gave their informed consent to take part in the study. The study was approved by the Ethics Committee of the University of Modena and Reggio Emilia. Table 1 shows demographic, clinical and neuropsychological data of PD patients included in the study.

Stimuli and experimental design

The Facial Emotion Recognition Battery used face pictures from the Ekman and Friesen [46] series showing the following expressions: neutral, happiness, sadness, disgust, fear and anger. The Battery included the following tasks:

1. Face Matching (FM) is a control task consisting of 14 trials. It requires selecting from a vertically arranged

Table 1 Demographic, clinical, and neuropsychological data of PD patients

Patient	Sex	Age	Disease duration (years)	More affected side	UPDRS	H.Y.	MMSE	BDI	MOCQ/R	FAB	Raven	MCST*
C.S.	M	73	5	left	40	2	26.45	15	12.00 PM	14	4	2%–16%
F.G.	M	67	02-mar	left	23	1	28.15	6	5.00 PM	16	4	5%–57%
L.R.	M	73	9	left	6	1	29.88	11	6.00 PM	15	3	5%–83%
C.E.	M	65	15	right	20	2	30.5	3	3.00 PM	15	2	6%–0%
M.C.	F	72	4	right	44	2.5	27.12	12	6.00 PM	14	4	6%–0%
C.R.	M	66	7	right	52	2	23.1	2	13 p	13	2	4%–50%
C.A.	M	68	3	left	37	3	25.2	14	7.00 PM	13	4	6%–16%
M.E.	F	70	7	right	27	1	27.3	5	2.00 PM	15	3	5%–28%
M.N.	F	60	5	right	37	2	25.8	8	12.00 PM	12	3	4%–31%
B.G.	M	56	7	left	21	1	26.9	9	5.00 PM	15	4	6%–12%
M.R.	F	70	7	right	21	2.5	25.3	11	9.00 PM	15	4	4%–29%
Z.G.	M	60	2	left	18	1	30.8	1	2.00 PM	13	4	6%–0%
R.B.	F	64	8	left	33	2.5	27	16	2.00 PM	15	3	4%–28%
P.N.	F	70	7	left	44	3	25.3	18	1.00 PM	10	4	4%–59%
G.R.	F	76	2	left	34	2.5	25.12	15	12.00 PM	13	4	4%–20%
B.C.	M	68	10	right	40	2	31.2	19	4.00 PM	16	3	4%–58%
B.A.	F	68	2	right	19	2	27.2	7	1.00 PM	14	4	6%–66.7%
F.V.	M	75	10	right	26	2	27.03	5	3.00 PM	16	4	6%–100%
F.A.	M	75	3	right	23	2	28.55	4	3.00 PM	13	4	5%–33.3%
F.V.	F	72	7	left	20	2	27.4	9	6.00 PM	11	2	1%–8%
G.G.	F	65	6	right	42	3	30.05	16	5.00 PM	17	3	6%–50%
M.A.	M	71	6	left	30	2	27.78	7	3.00 PM	11	2	2%–37.5%
M.E.	M	72	4	right	28	2	29.88	12	1.00 PM	18	4	6%–66.7%
S.G.	M	59	2	left	18	1.5	29.56	8	1.00 PM	13	4	4%–25%
S.M.	F	53	1	left	26	1	29.31	12	6.00 PM	18	4	6%–0%
T.I.	M	68	13	right	21	1.5	25.2	9	4.00 PM	15	3	4%–41.2%
V.M.	F	51	6	N.A.	N.A.	N.A.	28.78	7	12.00 PM	18	4	6%–4%

MMSE, Mini Mental State [43]; *BDI*, Beck Depression Inventory [44]; *MOCQ/R*, Maudsley Obsessive-Compulsive Questionnaire [45]; *MCST*, Modified Card Sorting Test; H.E. Nelson, 1976 [64]; *FAB*, Frontal Assessment Battery [65]; *UPDRS*, Unified Parkinson's Disease Rating Scale [66]; *H.Y.*, Hoehn & Yahr Scale [67]. Scores are corrected for age and education (MMSE, FAB) and age, sex and education (Raven).

* Categories achieved and perseverative errors

set of four faces with neutral expression the identical photograph of the stimulus face. Distracters are photographs of different persons of the same sex.
2. Facial Identity Recognition (FIR) measures the ability of recognising one person's identity despite different expressions. It consists of 14 trials. The subject is requested to select the target person among a vertically arranged set of four faces with different expressions.
3. Facial Affect Naming (FAN) requires the subject to select the name of the emotional expression of a face among five alternatives printed vertically below the stimulus face. The test consists of 25 trials.
4. Facial Affect Selection (FAS) requires the participant to select from a vertically arranged set of five faces the one that bears the expression corresponding to a target label. The test consists of 25 trials.
5. Facial Affect Matching (FAM) requires the subject to select among a vertically arranged set of five faces the one bearing the same expression as the stimulus face. The person making the stimulus and target expression are never the same and there is always one identity foil, i.e. a photograph of the same person of the target face with a different expression. The test consists of 25 trials.

The Emotional Prosody Recognition Battery consisted in a visual and auditory presentation of Italian sentences arranged in four different tasks:

1. Vocal Identity Discrimination (VID) requires indicating whether two sentences were pronounced by the same individual. The test consists of 16 pairs of neutral (aprosodic) stimuli.
2. Prosodic Discrimination (PrD) requires indicating whether two sentences presented were pronounced with the same prosodic intonation. The test consists of 16 pairs of sentences expressing four different intonations: interrogative, declaratory, exclamatory, and imperative.
3. Prosodic Affect Naming (PrAN) requires the subject to select among five alternatives the emotion conveyed by the intonation of a sentence. The five alternatives are presented on the computer screen. The test consists of 25 trials, 5 for each emotion (happiness, fear, disgust, anger, and sadness).
4. Prosodic Affect Discrimination (PrAD) requires indicating whether two sentences presented were pronounced with the same emotional prosody. It consists of 45 pair of sentences.

Only 11 out of 27 patients (7 right-sided and 4 left-sided) performed the Emotional Prosody Recognition Battery.

Results

Emotion recognition battery

The proportion of correct responses of PD patients and controls was compared for each of the different subtests by means of separate non-parametric analyses (Mann-Whitney U tests). Level of statistical significant was adjusted for multiple comparison with Bonferroni correction (alpha= 0.0102 for each test).

PD patients compared with controls, irrespectively of the most affected side, were significantly impaired at the Facial Affect Name Recognition (U=538.0; $p<0.001$; mean correct responses = 81.04% in PD patients and 89.35% in controls), and at the Facial Affect Matching tasks (U=450.5; $p<0.001$; mean correct responses = 72.59% in PD patients and 87.06% in controls; Fig. 1). PD patient impairment was close to significance at the Facial Affect Selection (U=628; $p=0.016$; mean correct

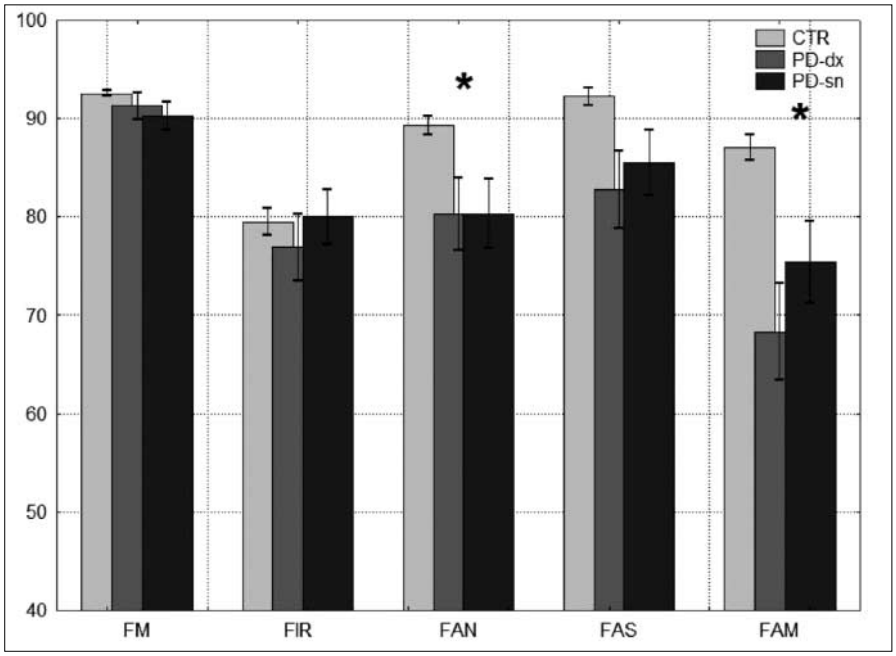

Fig. 1 Mean correct identification rate in the Emotion Recognition Battery across the two experimental groups. * represent statistical significance at $p= 0.0102$; whiskers are SE

responses = 84.74% in PD patients and 92.23% in controls). There was no significant difference between PD patients and controls at the Face Matching and at the Facial Identity Recognition tasks.

We then considered the proportion of responses for each of the five emotions at the Facial Affect Name Recognition. Adjusting the level of statistical significance for multiple comparisons with the Bonferroni correction (alpha=0.0102), PD turned out to be impaired on fear (U=582.5; p=0.005; mean correct responses = 56.29% in PD patients and 73. 82% in controls) and sadness recognition (U=608; p=0.01; mean correct responses = 79.26% in PD patients and 93.52% in controls), but they appeared to perform as well as control subjects with faces expressing happiness and there was no significant difference between PD patients and controls with faces expressing anger and disgust. In particular, with respect to controls, right-sided PD patients were impaired in recognition of fearful faces (U=225.5; p=0.004; mean correct responses = 50.77% in right-sided patients and 58.46% in left-sided patients) whereas left-side patients were impaired in recognition of sad faces (U=188; p<0.001; mean correct responses = 84.61% in right-sided patients and 72.31% in left-sided patients; Fig. 2).

Emotional prosody recognition battery

The proportion of correct responses of PD patients and controls was compared for each of the different subtests by means of separate non-parametric analyses (Mann-Whitney U tests). Level of statistical significance was adjusted for multiple comparison with Bonferroni correction (alpha=0.0127 for each test).

Compared with controls, PD patients were significantly impaired at the Vocal Identity Discrimination (U=198.5; p=0.003; mean correct responses = 69.79% in PD patients and 80.61% in controls), at the Prosodic Discrimination (U=153.5; p<0.001; mean correct responses = 73.96% in PD patients and 86.4% in controls) and at the Prosodic Affect Naming task (U=149.5; p<0.001; mean correct responses = 53% in PD patients and 77.53% in controls). There was no significant difference between PD patients and controls at the Prosodic Affect Discrimination task.

With respect to controls, right sided patients were significantly impaired at the Vocal Identity Discrimination (U=85.5; p=0.003; mean correct responses = 66. 96% in right-sided patients and 68.75% in left-sided patients), at the Prosodic Discrimination (U=93; p=0.006; mean correct responses = 73.21% in right-sided patients and 75% in left-sided patients) and at the Prosodic Affect Naming task (U=49.5; p<0.001; mean correct responses = 53.14% in right-sided patients and 55% in left-sided patients; Fig. 3).

Adjusting the level of statistical significance for multiple comparisons with the Bonferroni correction (alpha=0.0102), we considered the proportion of correct response for each of the five emotions at the Prosodic Affect Naming task. PD patients were impaired in recognising happy intonation (U=218; p=0.005; mean correct responses = 60% in PD patients and 88.25% in controls). In particular, right-sided PD patients were significantly impaired in recognising the intonation expressing disgust in comparison both with control subjects and with left-sided PD patients (U=67; p<0.001; mean correct responses = 11.43% in right-sided patients and 55% in left-sided patients Fig. 4) while the performance of left sided PD patients was significantly different from con-

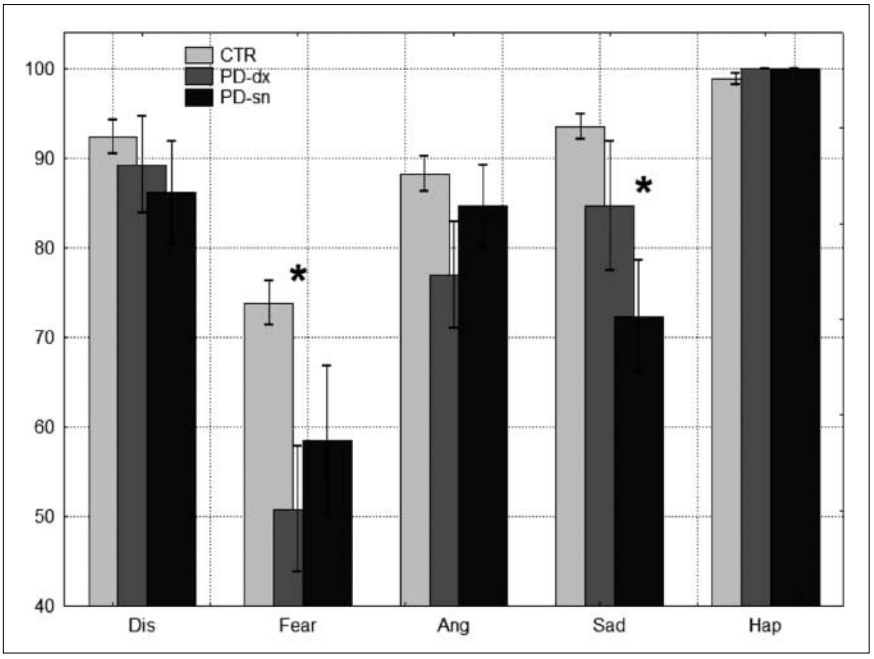

Fig. 2 Mean percentage of response for each of the five emotions to all images at the Facial Affect Name Recognition, at the Facial Affect Selection and at the Facial Affect Matching tasks of Emotion Recognition Battery. * represent statistical significance at p= 0.0102; whiskers are SE

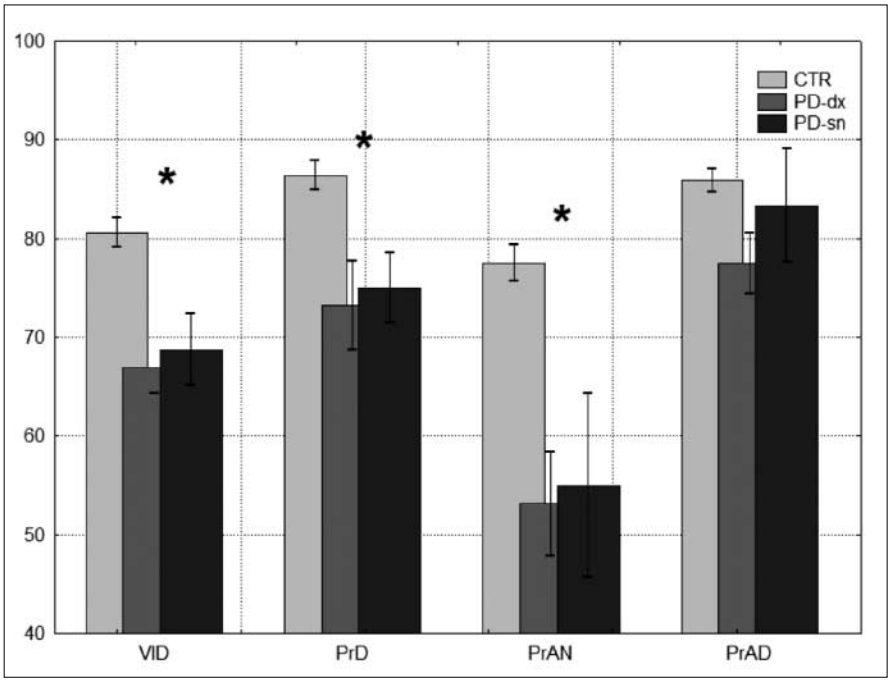

Fig. 3 Mean correct identification rate in the Emotional Prosody Recognition Battery across the two experimental groups. *represent statistical significance at p= 0.0127; whiskers are SE

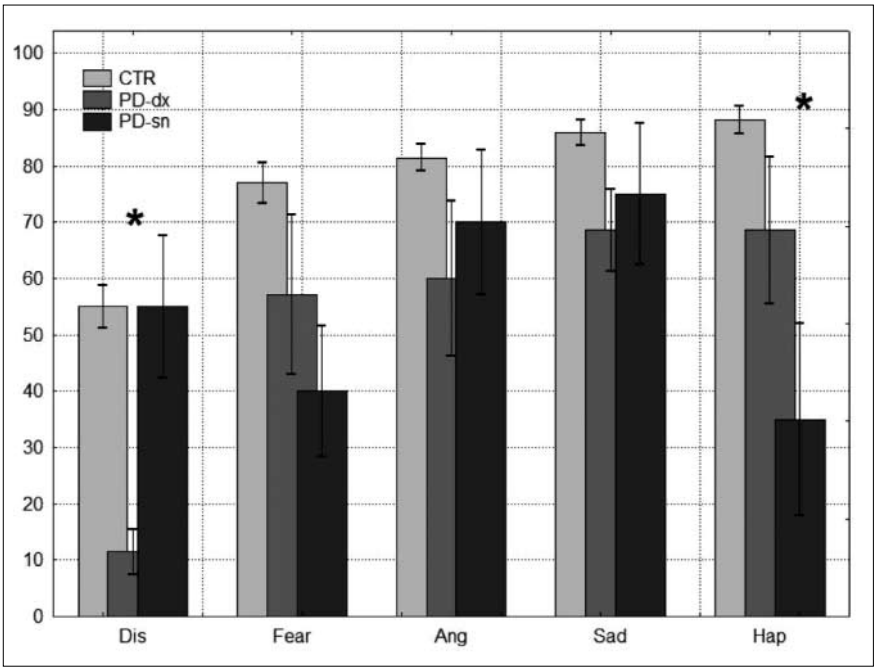

Fig. 4 Total percentages of correct response for each of the five emotions in the Emotional Prosody Recognition Battery across the two experimental groups. * represent statistical significance at p=0.0102; whiskers are SE

trols in recognising happy intonation (U=21; p=0.002; mean correct responses = 68.57% in right-sided patients and 35% in left-sided patients; Fig. 4).

Patients appeared to perform as well as control subjects in sadness, fear, and anger recognition.

Lastly, we did not find any significant correlation between the two emotional battery scores, the Beck Depression Inventory, the reduced version of the Maudsley Obsessional-Compulsive Questionnaire, tests exploring executive functions (FAB, Raven and MCST), the motor score at the UPDRS, and disease duration.

Conclusions

A number of studies have reported that PD patients are impaired in recognising emotional facial expressions [23–25, 35, 47] and emotional prosodic information [7, 35, 41, 42]. However, other neuropsychological studies failed in demonstrating differences between patients and controls in several emotional tasks [26, 31–33, 40].

These conflicting results may be attributed to selection bias (inclusion of patients with depression, cognitive impairment, different disease stages, medication, disease

duration and/or different most affected side). Also, it is possible that different findings depend on different tasks for assessing emotional processing,

With this study we have evaluated emotional processing across both the visual and the auditory domain in cognitively unaffected PD patients. The different tasks included in the two emotional batteries aimed at detecting generic or specific emotional deficit across the two sensory modalities.

The results of the Emotional Prosody Recognition Battery demonstrated that PD patients are impaired in processing not only emotional but also propositional prosody. This finding seems to parallel the aprosodic characteristic of speech produced by PD patients. In addition, the data from controls and patients confirmed that recognising emotions from prosody is more difficult than recognising emotions from facial expressions, and certain emotions such as disgust can be recognized only very poorly from prosody. Furthermore, only 11 out of 27 PD patients enrolled in this study were also examined with the Emotional Prosody Recognition Battery. For these reasons it is difficult to disentangle a possible emotional processing component from the mere general impairment in processing prosody. However, a more general impairment in processing prosodic intonation cannot explain the finding of a double dissociation between most affected side and most impaired emotion (with right-sided PD patients being impaired in processing intonation expressing disgust while left-sided PD patient are more impaired in processing happy intonation).

Emotion recognition impairment could not be explained by a more general cognitive deficit. Indeed, we excluded from this study cognitively impaired PD patients. In addition, those that were examined turned out to be unimpaired at the Face Discrimination, a test aimed at assessing perceptual processing of individual characteristics of faces. Moreover, lack of correlation between our tasks and the Beck Depression Inventory rules out the hypothesis that the emotion recognition impairment of PD patients can be attributed to depression.

UPDRS motor score did not correlate with the ability to recognize emotions from facial and prosodic cues, thus suggesting that the facial emotion recognition impairment does not simply follow the motor disease and is probably due to the damage of brain structures different from those causing the motor impairment.

The results of the Emotion Recognition Battery also suggest the possibility of a selective deficit in the facial expression recognition which depends on the side most affected by the disease, with right-sided PD patients being impaired in recognition of faces expressing fear whereas left-sided patients are impaired in recognition of sad expression. A number of clinical [48] and neuroimaging studies [49, 50] have shown the role of the amygdala in processing the facial expression of fear [51–53]. In particular, neuropsychological [54, 55] and neuroimaging studies [56–58] have consistently found that the right amygdala is involved in processing fearful facial expressions. Activation of the left amygdala in processing faces expressing sadness have also been found in normal subjects [59]. Moreover, morphological and functional abnormalities in left amygdala have been found in psychiatric diseases and in particular in depression [60], in schizophrenia [61] and in the Asperger syndrome [62, 63].

A number of studies have described mild executive dysfunction in patients with early Parkinson Disease and the role of impaired cortico-basal connections in determining them [68, 69]. However the fact that we did not find any correlation between tasks exploring frontal dysfunctions and scores at the two emotional batteries argues against ascribing the emotional deficit to impaired cortico-basal connections. In contrast, our results are in line with data indicating that the amygdala is damaged early during the course of Parkinson's disease. Braak et al. [1] suggested that Parkinson's disease is characterized by a progressive pathological process marked by the presence of α-synuclein-immunoreactive inclusions, affecting circumscribed regions of the brain in a topographically predictable sequence, during which components of the olfactory, autonomic, limbic, and somatomotor system become progressively involved.

According to this view, the amygdala (and particularly its central subnucleus) is damaged early during the course of the disease, possibly even before substantia nigra/pars compacta, which is responsible for the motor impairment in PD.

Evidence about performance at emotional processing tasks in atypical Parkinsonism is still lacking. However, it is possible that when carefully investigated, the impaired emotional processing of PD patients might turn out to be a useful complement in the differential diagnosis of Parkinsonian syndromes.

Acknowledgments The Authors thank Anna Magherini for her assistance in selecting patients and in administering the UPDRS scale to a subgroup of them.

References

1. Braak H, Bohl JR, Muller CM et al (2006) Stanley Fahn Lecture 2005: The staging procedure for the inclusion body pathology associated with sporadic Parkinson's disease reconsidered. Mov Disord 21:2042–2051
2. Adolphs R (2002) Neural systems for recognizing emotion. Curr Opin Neurobiol 12:169–177
3. Adolphs R, Damasio H, Tranel D (2002) Neural systems for recognition of emotional prosody: a 3-D lesion study. Emotion 2:23–51
4. Breitenstein C, Daum I, Ackermann H (1998) Emotional processing following cortical and subcortical brain damage: contribution of the fronto-striatal circuitry. Behav Neurol 11:29–42
5. Buchanan TW, Lutz K, Mirzazade S et al (2000) Recognition of emotional prosody and verbal components of spoken language: an fMRI study. Brain Res Cogn Brain Res 9:227–238

6. Hornak J, Rolls ET, Wade D (1996) Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal lobe damage. Neuropsychologia 34:247–261
7. Scott SK, Young AW,Calder AJ et al (1997) Impaired auditory recognition of fear and anger following bilateral amygdala lesions. Nature 385:254–257
8. Sprengelmeyer R, Young AW, Schroeder U et al (1999) Knowing no fear. Proc R Soc Lond B Biol Sci 266:2451–2456
9. Dolan RJ, Morris JS, de Gelder B (2001) Crossmodal binding of fear in voice and face. Proc Natl Acad Sci U S A 98: 10006–10010
10. Calder AJ, Lawrence AD, Young AW (2001) Neuropsychology of fear and loathing. Nat Rev Neurosci 2:352–363
11. Hennenlotter A, Schroeder U, Erhard P et al (2004) Neural correlates associated with impaired disgust processing in pre-symptomatic Huntington's disease. Brain 127:1446–1453
12. Gorno-Tempini ML, Pradelli S, Serafini M et al (2001) Explicit and incidental facial expression processing: an fMRI study. Neuroimage 14:465–473
13. Phillips ML, Young AW, Senior C et al (1997) A specific neural substrate for perceiving facial expressions of disgust. Nature 389:495–498
14. Sprengelmeyer R, Rausch M, Eysel UT, Przuntek H (1998) Neural structures associated with recognition of facial expressions of basic emotions. Proc R Soc Lond B Biol Sci 265:1927–1931
15. Sprengelmeyer R, Young AW, Calder AJ et al (1996) Loss of disgust. Perception of faces and emotions in Huntington's disease. Brain. 119:1647–1665
16. Sprengelmeyer R, Young AW, Sprengelmeyer A et al (1997) Recognition of facial expression: selective impairment of specific emotions in Huntington's disease. Cognitive Neuropsych 14:839–879
17. Calder AJ, Keane J, Manes F et al (2000) Impaired recognition and experience of disgust following brain injury. Nat Neurosci 3:1077–1078
18. Sprengelmeyer R, Schroeder U, Young AW, Epplen JT (2006) Disgust in pre-clinical Huntington's disease: a longitudinal study. Neuropsychologia 44:518–533
19. Gray JM, Young AW, Barker WA et al (1997) Impaired recognition of disgust in Huntington's disease gene carriers. Brain 120:2029–2038
20. Wang K, Hoosain R, Yang RM et al (2003) Impairment of recognition of disgust in Chinese with Huntington's or Wilson's disease. Neuropsychologia 41:527–537
21. Sprengelmeyer R, Young AW, Pundt I et al (1997) Disgust implicated in obsessive-compulsive disorder. Proc R Soc Lond B Biol Sci 264:1767–1773
22. Dimberg U (1982) Facial reactions to facial expressions. Psychophysiology 19:643–647
23. Jacobs DH, Shuren J, Bowers D, Heilman KM (1995) Emotional facial imagery, perception, and expression in Parkinson's disease. Neurology 45:1696–1702
24. Dujardin K, Blairy S, Defebvre L et al (2004) Deficits in decoding emotional facial expressions in Parkinson's disease. Neuropsychologia 42:239–250
25. Suzuki A, Hoshino T, Shigemasu K, Kawamura M (2006) Disgust-specific impairment of facial expression recognition in Parkinson's disease. Brain 129:707–717
26. Wieser MJ, Muhlberger A, Alpers GW et al (2006) Emotion processing in Parkinson's disease: dissociation between early neuronal processing and explicit ratings. Clin Neurophysiol 117:94–102
27. Tessitore A, Hariri AR, Fera F et al (2002) Dopamine modulates the response of the human amygdala: a study in Parkinson's disease. J Neurosci 22:9099–9103
28. Yoshimura N, Kawamura M, Masaoka Y, Homma I (2005) The amygdala of patients with Parkinson's disease is silent in response to fearful facial expressions. Neuroscience 131:523–534
29. Sprengelmeyer R, Young AW, Mahn K et al (2003) Facial expression recognition in people with medicated and unmedicated Parkinson's disease. Neuropsychologia 41:1047–1057
30. Salgado-Pineda P, Delaveau P, Blin O, Nieoullon A (2005) Dopaminergic contribution to the regulation of emotional perception. Clin Neuropharmacol 28:228–237
31. Adolphs R, Schul R, Tranel D (1998) Intact recognition of facial emotion in Parkinson's disease. Neuropsychology 12:253–258
32. Smith MC, Smith MK, Ellgring H (1996) Spontaneous and posed facial expression in Parkinson's Disease. J Int Neuropsychol Soc 2:383–391
33. Pell MD, Leonard CL (2005) Facial expression decoding in early Parkinson's disease. Brain Res Cogn Brain Res 23:327–340
34. Heilman KM, Bowers D, Speedie L, Coslett HB (1984) Comprehension of affective and nonaffective prosody. Neurology 34:917–921
35. Blonder LX, Gur RE, Gur RC (1989) The effects of right and left hemiparkinsonism on prosody. Brain Lang 36:193–207
36. Benke T, Bosch S, Andree B (1998) A study of emotional processing in Parkinson's disease. Brain Cogn 38:36–52
37. Crucian GP, Huang L, Barrett AM et al (2001) Emotional conversations in Parkinson's disease. Neurology 56:159–165
38. Pell MD (1996) On the receptive prosodic loss in Parkinson's disease. Cortex 32:693–704
39. Pell MD, Leonard CL (2003) Processing emotional tone from speech in Parkinson's disease: a role for the basal ganglia. Cogn Affect Behav Neurosci 3:275–288
40. Caekebeke JF, Jennekens-Schinkel A, van der Linden MA et al (1991) The interpretation of dysprosody in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 54:145–148
41. Kan Y, Kawamura M, Hasegawa Y et al (2002) Recognition of emotion from facial, prosodic and written verbal stimuli in Parkinson's disease. Cortex 38:623–630
42. Yip JT, Lee TM, Ho SL et al (2003) Emotion recognition in patients with idiopathic Parkinson's disease. Mov Disord 18:1115–1122
43. Folstein MF, Folstein SE, McHugh PR (1975) "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 12:189–198
44. Beck AT, Ward CH, Mendelson N (1961) An inventory for measuring depression. Arch Gen Psychiatry 4:561–571
45. Sanavio E, Bertolotti G, Michielin P et al (1986) CBA-2.0 Scale Primarie. Firenze: Organizzazioni Speciali
46. Ekman P, Friesen WV (1976) Pictures of facial affect. Palo Alto: Consulting Psychologist Press
47. Mayeux R, Stern Y,Rosen J, Levental J (1981) Depression, intellectual impairment and Parkinson's disease. Neurology 31:645–650
48. Broks P, Young AW, Maratos EJ et al (1998) Face processing impairments after encephalitis: amygdala damage and recognition of fear. Neuropsychologia 36:59–70
49. Morris JS, Ohman A, Dolan RJ (1998) Conscious and unconscious emotional learning in the human amygdala. Nature 393:467–470
50. Whalen PJ, Bush G, McNally RJ et al (1998) The emotional counting Stroop paradigm: a functional magnetic resonance imaging probe of the anterior cingulate affective division. Biol Psychiatry 18:1219–1228
51. Wright CI, Fischer H, Whalen PJ et al (2001) Differential prefrontal cortex and amygdala habituation to repeatedly presented emotional stimuli. Neuroreport 12:379–383
52. Rolls ET (2000) The orbitofrontal cortex and reward. Cereb Cortex 10:284–294

53. Rolls ET (2007) The representation of information about faces in the temporal and frontal lobes. Neuropsychologia 45:124–143
54. Meletti S, Benuzzi F, Rubboli G et al (2003) Impaired facial emotion recognition in early-onset right mesial temporal lobe epilepsy. Neurology 60:426–431
55. Sanz-Martin A, Guevara MA, Corsi-Cabrera M et al (2006) Efecto diferenmcial de la lobectomía temporal izquierda y derecha sobre el reconocimiento y la experiencia emocional en pacientes con epilepsia. 42:391–398
56. Morris JS, Friston KJ, Buchel C et al (1998) A neuromodulatory role for the human amygdala in processing emotional facial expressions. Brain 121:47–57
57. Morris JS, Ohman A, Dolan RJ (1999) A subcortical pathway to the right amygdala mediating unseen fear. Proc Natl Acad Sci U S A 96:1680–4685
58. Whalen PJ, Rauch SL, Etcoff NL et al (1998) Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. J Neurosci 18:411–418
59. Blair RJ, Morris JS, Frith CD et al (1999) Dissociable neural responses to facial expressions of sadness and anger. Brain 122:883–893
60. Surguladze S, Brammer MJ, Keedwell P et al (2005) A differential pattern of neural response toward sad versus happy facial expressions in major depressive disorder. Biol Psychiatry 57:201–209
61. Kucharska-Pietura K, Russell T, Masiak M (2003) Perception of negative affect in schizophrenia–functional and structural changes in the amygdala. Review. Ann Univ Mariae Curie Sklodowska [Med] 58:453–458
62. Baron-Cohen S, Ring HA, Bullmore ET et al (2000) The amygdala theory of autism. Neurosci Biobehav Rev 24:355–364
63. Baron-Cohen S, Ring HA, Wheelwright S et al (1999) Social intelligence in the normal and autistic brain: an fMRI study. Eur J Neurosci 11:1891–1898
64. Nelson HE (1976) A modified card sorting test sensitive to frontal lobe defects. Cortex 12:313–324
65. Dubois B, Slachevsky A, Litvan I, Pillon B (2000) The FAB: a Frontal Assessment Battery at bedside. Neurology 55:1621–1626
66. Fahn S, Elton RL and M.o.t.U.D. Committee (1987) The Unified Parkinson's Disease Rating Scale. In: Fahn S, Marsden CD, Calne DB, Goldstein M (eds) Recent developments in Parkinson's disease. Macmillan Health Care Information: Florham Park, NJ
67. Hoehn M, Yahr MD (1967) Parkinsonism: Onset, progression and mortality. Neurology 17:427–442
68. Levin BE, Katzen HL (2005) Early cognitive changes and nondementing behavioral abnormalities in Parkinson's disease. Adv Neurol 96:84–94
69. Zgaljardic DJ, Foldi NS, Borod JC (2004) Cognitive and behavioral dysfunction in Parkinson's disease: neurochemical and clinicopathological contributions. J Neural Transm 111:1287–1301